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PRINTED IN GREAT BRITAIN

ASSESSMENTS OF LOSSES IN SUGAR BEET DUE TO VIRUS YELLOWS IN GREAT BRITAIN, 1942-52

by R. HULL

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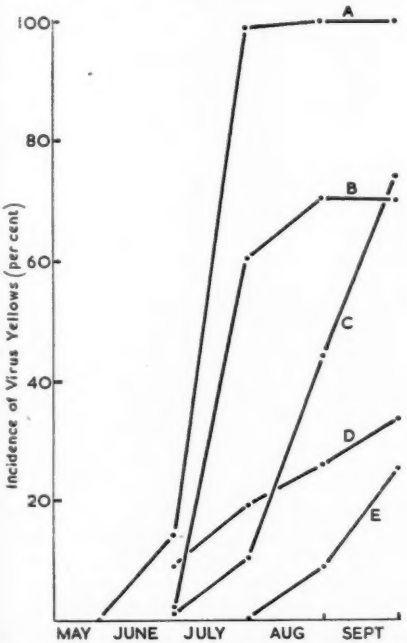
Records of the incidence of Virus Yellows in sugar beet crops have been made each year since 1942 as part of a comprehensive survey of the incidence of sugar beet diseases in Great Britain. The survey is made by the agricultural staff of the British Sugar Corporation, and is organized from Dunholme Field Station where the reports are collated each month and the results circulated to those taking part in the survey. Instruction in disease recognition and methods of counting has been given to the sugar factory agriculturists and fieldmen by means of lectures, field courses and circulars. The present system of quantitative reports was started on a small scale in 1942 and was extended in 1946 to all of the Sugar Corporation's seventeen factory areas in England, to the southern area covering the south of England and to the Scottish beet growing area centred on the factory at Cupar, Fifeshire.

Each fieldman, whose area contains between 1,000 and 4,000 acres of sugar beet, counts diseased plants at the end of each month from May to September in one field of about 10 acres. The number of fields thus sampled each year



A. Progress of the disease when infection appears early and conditions are favourable for rapid spread. (Spalding, 1949)

B. and D. Severe and potentially severe outbreaks restricted by unfavourable conditions for spread during the summer.
(B. Bury St. Edmunds, 1948)
(D. Moulsford, Berks, 1950)

C. and E. Infection spreading rapidly only towards the end of the season.
(C. Spalding, 1952)
(E. Spalding, 1951)

Fig. 1. Types of curves obtained for the spread of Virus Yellows under differing seasonal conditions.

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varies between 135 and 155. The distribution of the sample fields roughly corresponds with the density of the sugar beet crop in the various parts of the country. As far as possible a field is chosen each year belonging to the same farmer, one who is a consistent grower and whose crops are judged to be representative of the area. The number of plants with Yellows is counted in ten samples of 100 plants spaced at equal intervals along the diagonals of the field. The yield loss by Virus Yellows is directly proportional to the length of time a plant is infected (Watson, Watson and Hull, 1946), thus periodical counts of the number of infected plants in a crop measure the severity of infection. The variations in the rate of spread of the disease in fields are illustrated in Fig. 1.

The curves for the average percentage infection in all the fields in which counts have been made are shown in Fig. 2. Those for 1942–44 are the average for 18 fields located in the areas of the Brigg and Bardney factories in Lincolnshire and the Kelham factory in Nottinghamshire ; in 1945 a further five fields in the Peterborough factory area were included. The curves for 1946–52 are for the 150 or so sample fields distributed in all beet growing areas. More detailed results for the different parts of the country for the years 1946–49 have been published previously (Hull, 1950).

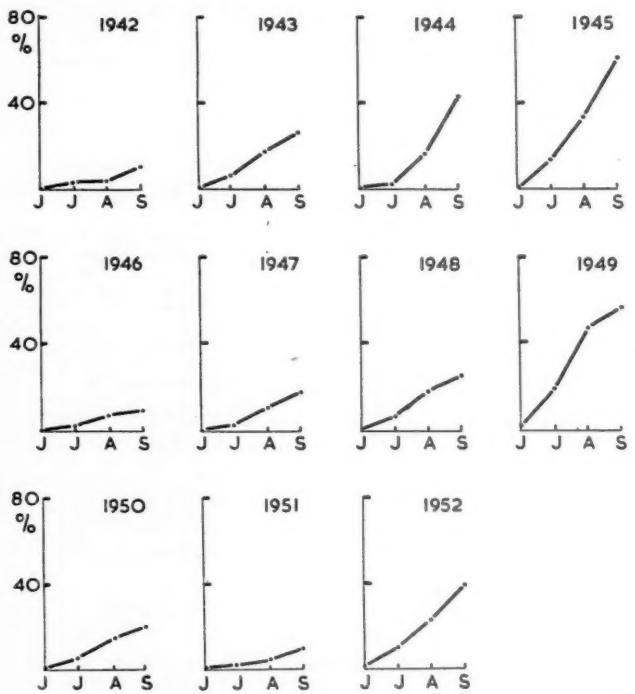

Fig. 2. Average percentage infection of sugar beet crops by Virus Yellows at the end of each month from June to September, 1942–45, in east Midlands, and from 1946–52 throughout Great Britain.

There was a gradual build-up of the disease over four years from a very low level of infection in 1942 and 1946 to the peak infections in 1945 and 1949. The lowest recorded level of infection was in 1951, but in 1952 very heavy infestations of both green and black aphids early in the summer resulted in a widespread, though rather late, development of the disease.

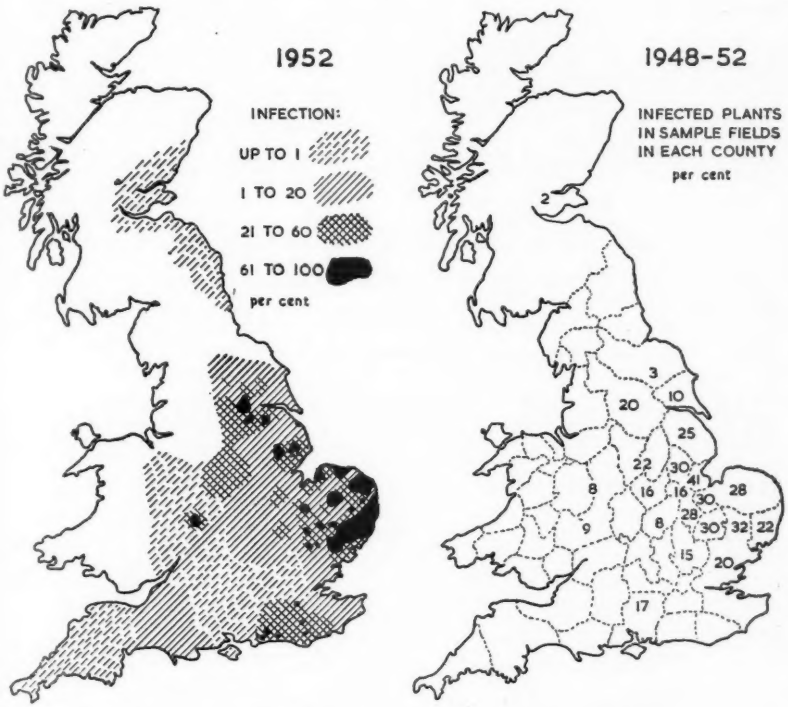

Fig. 3. Incidence of Virus Yellows in sugar beet crops at the end of August.

The fields on which the assessments are made each year have an aggregate area of 1,500 acres—a very small sample of the whole crop, which is about 400,000 acres. To check the accuracy of the results, the incidence of Virus Yellows is independently estimated at the end of August. The factory agriculturists assess, from the reports of the fieldmen and from their own observations, the acreage in which infection falls within each of four categories : less than 1 per cent ; 1 to 20 per cent ; 21 to 60 per cent ; and 61 to 100 per cent. From these data the mean percentage infection is calculated for each factory area by taking the mean percentage infection of the acreage in each category to be : 1 per cent ; 10 per cent ; 40 per cent ; and 80 per cent, respectively. The mean infection over the whole country is calculated in the same way from the sum of the acreage falling in each category in all areas as a proportion of the total acreage for the country. The results (Table 1) agree reasonably well with those obtained from the sample field counts.

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TABLE 1

COMPARISON OF ESTIMATES OF THE MEAN PERCENTAGE INFECTION OF SUGAR BEET WITH VIRUS YELLOWS AT THE END OF AUGUST IN GREAT BRITAIN

Method	1946	1947	1948	1949	1950	1951	1952
Acreage estimates	7·1	12·5	17·5	46·1	16·2	5·6	25·1
Sample field counts	6·0	9·5	17·9	47·6	13·7	3·5	22·0

In 1952, the agriculturists marked the areas where crops showed infection within each of the four categories on maps having a scale of four miles to one inch. A simplified map, prepared from these originals for Great Britain at the end of August 1952, is given on the left of Fig. 3. Against this, on the right, is a map showing the mean percentage for 1948–52 of infected plants found by counts in the sample fields in each county, also at the end of August.

ESTIMATES OF THE LOSSES CAUSED BY VIRUS YELLOWS

The loss caused by the disease on artificially infected experimental plots has been shown to be 4–5 per cent of the sugar yields for every week during which plants show Yellows symptoms until harvested (Watson, Watson and Hull, 1946). This result may be used in conjunction with the sample field counts to assess the annual loss caused by the disease. The length of time the plants have been infected is taken as the number of weeks between the observation date and the middle of October. Thus the increase in percentage count at the end of June, July, August and September over that of the previous count is taken as the proportion of plants showing symptoms for 15, 11, 7 and 2 weeks respectively. The product of the proportion of freshly infected plants for each month and the number of weeks to harvest is then multiplied by the experimentally determined figure of 4–5 per cent to give the percentage loss of sugar yield for the infections developing during that month. The sum of these percentage losses for each of the four months is the total percentage loss of sugar yield, which has been calculated assuming both 4 per cent and 5 per cent loss of sugar yield for each week of infection (Table 2).

TABLE 2

YIELD OF SUGAR BEET IN GREAT BRITAIN AND THE CALCULATED LOSSES CAUSED BY VIRUS YELLOWS

Year	Calculated Percentage Loss of Sugar Yield			Actual Yield per Acre		Estimated Potential Yield of Roots* per Acre	Estimated Loss of Root Yield
	A†	B†	C†	Sugar	Roots*		
				tons	*tons*	*tons*	*tons (000)*
1945	—	—	13·9	1·57	9·81	—	—
1946	2·3	2·9	4·7	1·74	10·87	11·16	120
1947	3·8	4·5	9·6	1·44	8·98	9·36	141
1948	6·5	7·9	10·8	1·77	11·07	11·94	347
1949	17·4	21·8	27·1	1·51	9·44	11·80	944
1950	5·0	6·2	8·5	2·12	12·23	14·01	317
1951	1·5	2·1	3·6	1·85	11·56	11·78	90
1952	8·9	11·1	14·6	1·79	11·16	12·39	486

* Converted to equivalent yields at a sugar content of 16 per cent.

† A. Assuming 4 per cent loss for each week infected. B. Assuming 5 per cent loss for each week infected.
C. Assuming 2·4 cwt. loss for each 10 per cent infection at the end of August.

An estimate by another method is obviously desirable because of the sampling errors of the disease assessment and the uncertainty whether the results of a few infection experiments are applicable to the whole of the country's crop. The check method used has been to calculate a regression of each factory's annual sugar yield (product of mean yield of roots and mean sugar percentage, as recorded by the British Sugar Corporation) on the mean Virus Yellows infection at the end of August, assessed from the acreage infected estimate.

This regression coefficient is highly significant and shows that there is a loss of about $2\frac{1}{4}$ cwt. per acre of sugar yield for every 10 per cent Yellows infection recorded at the end of August. The method is open to the criticism that some factor which influences the incidence of Virus Yellows may also have an adverse effect on yield ; for instance, Virus Yellows may be worse in dry years when the crop is suffering from drought, or in years when sowing is late. When the effect of these two factors is eliminated by calculating a multiple regression of sugar yield on (*a*) rainfall during the cropping season, (*b*) mean sowing date, and (*c*) Virus Yellows infection, the regression coefficient for the latter is altered very little. The partial regression coefficient of sugar yield in tons per acre for every 1 per cent of plants with Yellows infection at the end of August, calculated from the data for the years 1946–52, is :

$$b = 0{\cdot}01218 \pm 0{\cdot}001904 \ (t = 6{\cdot}40)$$

This regression coefficient multiplied by the mean percentage of plants with Yellows gives the estimated percentage loss of sugar yield (Table 2). The losses calculated from the regression coefficient are higher than those calculated by the previous method, even when a weekly loss of 5 per cent is assumed. It seems probable that some other factor must be responsible for this large regression, for it is unlikely that 5 per cent is an underestimate of the weekly loss of sugar yield. Numerous infection experiments made in recent years have confirmed that plots which are yellow in July yield about one-half as much sugar as those that remain healthy until harvest. This may be compared with the calculated loss of 49·5 per cent for infected plants counted at the end of July, assuming 4·5 per cent loss of sugar yield for each week of infection. Thus 4·5 per cent per week is adjudged to be a reasonable figure on which to base calculations of losses, and the regression suggests that it is unlikely to be an unduly high estimate. The expected yield of roots for each year, had there been no Yellows, has been calculated using this figure, and in conjunction with the figures for the total sugar beet acreage in Great Britain it has been used to calculate the tonnage lost by the disease (Table 2).

I wish to express appreciation of the work of the agricultural staff of the British Sugar Corporation, Ltd. in making this survey, and to thank Mr. J. D. H. Dunwoody and members of the Statistical Department at Rothamsted for calculating the regression and giving much helpful advice from time to time on procedure.

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HOT-WATER TREATMENT OF STRAWBERRY RUNNERS

by L. N. STANILAND

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Strawberries are attacked by the leaf and bud eelworms, *Aphelenchoides fragariae* (Bos) and *A. ritzema-bosi* (Schwartz) in all strawberry-growing districts. In some districts, notably the Cheddar Valley, strawberries also suffer from attacks by the Stem and Bulb eelworm, *Ditylenchus dipsaci* (Kühn). These pests may be controlled by hot-water treatment (Staniland, 1933 ; Hodson, 1934), but this has never become a regular practice, as the usual method of immersing the runners for 20 minutes in water at 110° F., followed by a plunge into cold water, has often caused damage, especially when planting conditions have been unfavourable.

In the course of unpublished work carried out as far back as 1926, I found that good results could be obtained by the treatment of strawberry runners for 9 minutes at 113° F., and in subsequent work with D. R. Barber at Seale Hayne Agricultural College in 1936 (also unpublished) a study was made of the time necessary for the runners to heat through to the temperature of the bath. The present article reviews part of this past work, and gives results obtained in investigations re-opened in 1949 on variations of the hot-water treatment to secure effective control of eelworm, especially *Ditylenchus dipsaci*, with reduced risk of injury to the runners. An improved bath is also described.

HEAT ABSORPTION BY STRAWBERRY RUNNERS

The method used in 1936 for the determination of the rate of penetration of heat into the tissues of strawberry runners was similar to that used on work in the heat treatment of bulbs (Staniland and Barber, 1937). The temperature of the growing-point region in the crown of the runner was recorded by means of a pair of differential thermocouples mounted in a suitable holder and used in conjunction with a low-resistance moving coil galvanometer. The apparatus is shown in Fig 1. Two copper-nickel thermocouples were made from 50 S.W.G. wire, the nickel wire being bare and the copper wire enamelled. The wires were spot-welded at their junctions and enclosed in bent hypodermic needles, sealed at their ends. One needle was inserted into the plant material so that the point reached to the depth desired. The other needle, used as a standardizing couple, was maintained at the temperature of the water. The apparatus was mounted on vertical staging, with a rack and pinion for lowering it into the bath.

The strawberry runner crowns were severed about half an inch below the growing point region, and the remainder of the crown, including the larger leaves and roots, was rejected. The apparatus was then lowered into a constant-temperature water bath, fitted with a mechanical stirrer, so that the cut surface of the crown was just exposed above the water level. An observation was then made of the time taken for the deflection of the galvanometer to fall to zero, by which time the internal temperature at the tip of the needle within the crown was the same as that of the water. The crown was then removed from the needle and cut through longitudinally ; if it was then found that the needle had not penetrated to the growing region, the result was rejected.

Over 60 determinations were made on runners of various sizes. It was found that the majority of the larger runners heated through to the temperature of the water in 3–4 minutes ; smaller runners heated through in 2–3 minutes.

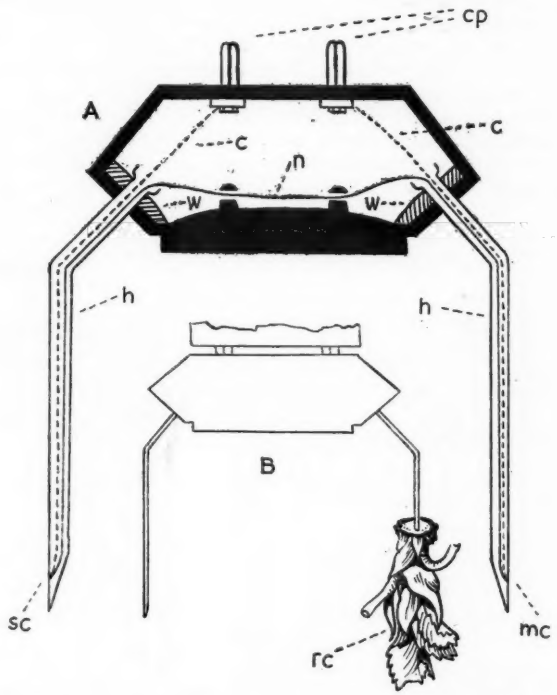

Fig. 1. Construction and use of differential thermocouple for determining temperature in strawberry runners.
cp. contact pin for connection to galvanometer. c. copper wire. n. nickel wire. h. hypodermic needle. w. hard wax seal. sc. standardizing couple. mc. measuring couple. rc. runner crown. A shows apparatus in section, B method of use.

TIME REQUIRED TO KILL EELWORMS

Determinations of the time required to kill eelworms in the runners were made in the laboratory during 1950, using a simple bath constructed from a 5-gallon oil drum lagged with carpet felt, with a wire cage for the plants and heated by a bunsen burner regulated by means of a screw-operated pinch tap. With occasional hand agitation of the water, the temperature of this bath could be kept constant within 0·25° F.

As excellent results had been obtained by treating chrysanthemum stools for 5 minutes at 115° F. instead of 20–30 minutes at 110° F. (Staniland, 1950), and by treating strawberry runners for 9 minutes at 113° F. in the experiments in 1926, it was decided to try a temperature of 115° F. in the re-opened investigations

on strawberry runners. It was found that at this temperature the two species of *Aphelenchoides* and active *D. dipsaci* were killed in 4 minutes. Since it took only 3–4 minutes for the runners to heat through to the temperature of the bath, it seemed that a total immersion period of from 7 to 8 minutes should be sufficient, as the *Aphelenchoides* species are ectoparasitic and *D. dipsaci* does not appear to penetrate deeply into the crown tissues. To test this supposition, a number of heavily infested maiden plants approximating in size to large runners were treated at 115° F. for periods of 7 and 10 minutes, followed by a cold water plunge. Examination of the treated plants by the Baermann funnel method showed that a complete kill of *D. dipsaci* was obtained in both treatments, but that a few *Aphelenchoides* survived the 7-minute immersion, possibly because there were a few dried individuals present on a feeding area, and such eelworms are more resistant to heat.

AN IMPROVED HOT-WATER BATH

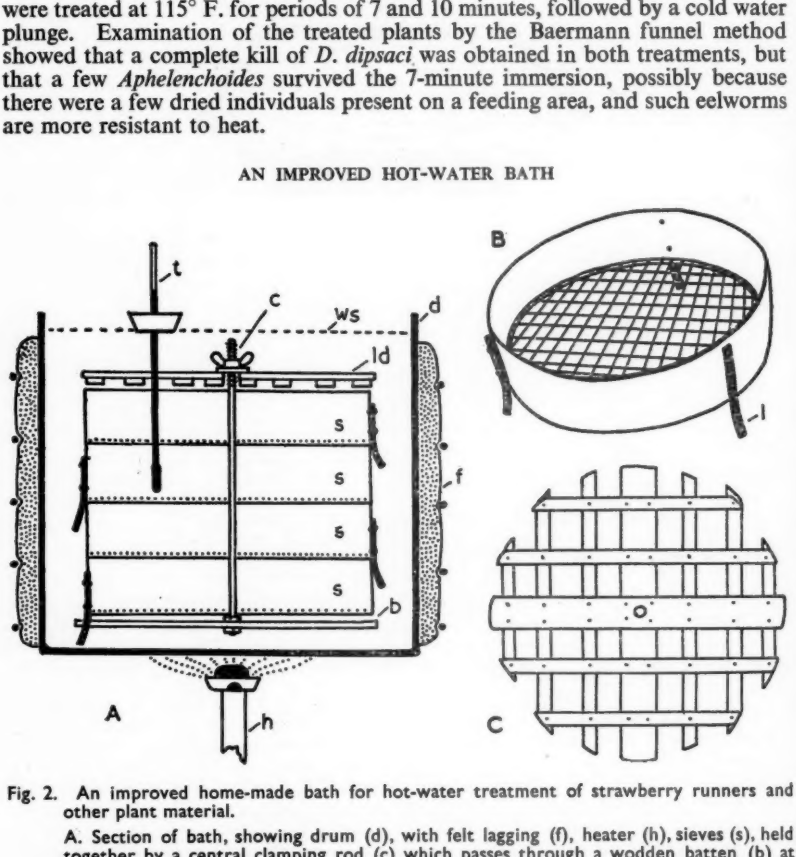

Fig. 2. An improved home-made bath for hot-water treatment of strawberry runners and other plant material.

A. Section of bath, showing drum (d), with felt lagging (f), heater (h), sieves (s), held together by a central clamping rod (c) which passes through a wodden batten (b) at the base, and a lath lid (ld) on top. A thermometer (t) is pushed through a cork floating on the water surface (ws).

B. Detail of sieve, showing three metal legs (l) bent out slightly at the base.

C. Lath lid for top sieve, with central hole for clamping bolt.

For testing the treatments on a field scale, a larger bath was required. The apparatus used was similar to that described in the Ministry's leaflet, *The Hot-water Treatment of Plants**, with some recent improvements as shown

* Available free from the Ministry of Agriculture (Publications), 36 Chester Terrace, Regent's Park, London, N.W.1.

in Fig 2. It consisted of a galvanized-iron water drum with a capacity of about 60 gallons, lagged on the outside with felt, and heated by a gas-ring or paraffin-vapour stove. The runners were placed for treatment into four sieves, each about 4 inches smaller in diameter than the drum, held together by a central clamping bolt passing through a batten at the bottom and a lath lid at the top. In use the drum contained some 40 gallons of water, and some 250 runners could be treated at a time. It was found that if the initial temperature of the water was brought to 117° F., it would drop almost exactly to 115° F. when the charge of washed runners was put in. After the assembly of sieves had been lowered into the bath, it was raised and lowered several times under the water to effect agitation and distribute the heat evenly among the runners. When the temperature did not quite drop to 115° F., the sieves were raised out of the water, and the drippings were allowed to run back before lowering them again : this quickly lowered the temperature by about half a degree. When necessary, the heater was placed under the bath during the treatment. In practice, no difficulty was found in maintaining a steady temperature of 115° F. for only 7 minutes.

On average, the time taken between the loading of one charge of runners and the next, including the cold plunge, was 15 minutes, so that the rate of treatment was 1,000 runners per hour—rather faster than they could be planted by two men.

FIELD TRIALS WITH TREATED RUNNERS

To observe the effect of the treatment on the growth of healthy runners, small batches of the variety Axbridge Early were treated at Draycott in Somerset on July 27, 1949 for 7, 10 and 15 minutes at 115° F., for comparison with others treated for 30 minutes at 110° F. and with untreated controls. The runners were planted on the day of treatment in a moist soil and were well firmed in. On August 4 it was found that the plants treated for 7 and 10 minutes were not suffering any ill-effects from the treatment, and those treated for 15 minutes showed only slight marginal scorching of the leaves. Examination of the root systems showed that the delay in forming new roots was only about 2–3 days, but it was a little longer in those treated for 15 minutes. Later examinations showed that the subsequent root growth was satisfactory in all the plants treated at 115° F., and that it was as good as that of the untreated controls. The treatments for 30 minutes at 110° F. resulted in much poorer root growth. Similar trials on batches of healthy Royal Sovereign runners, treated on August 2, 1949, gave much the same results as those on the Axbridge Early.

The first bulk treatment of runners from a stock heavily infested with eelworm was carried out at Cheddar on August 5, 1949. Here, nearly 5,000 runners were taken from a bed of Royal Sovereign heavily infested with the Stem eelworm, *Ditylenchus dipsaci*. About half the runners exhibited definite symptoms of attack and many were severely stunted. *Aphelenchoides* spp. were also present, but to a less extent. The runners were washed, treated for 7 minutes at 115° F., and given a plunge into cold water immediately they were lifted from the bath. Some of the runners were planted on the same day, but the weather was so hot that the planters found it difficult to carry on with the work ; the rest of the treated runners were therefore covered with damp sacks and kept in a cool shed until they were planted on August 6 and 7. The soil was exceptionally dry, and it was partially moistened before planting by means of a sprinkler. The plants were well firmed in. A count made on August 19 showed that the loss of plants was only 1 per cent. Rooting appeared to have

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been delayed slightly, but, by that time, it was prolific and healthy, and the plants showed practically no damage apart from slight edge scorch on a few outside leaves. The plants were then examined at intervals over a period of two years. No symptoms of Stem eelworm attack were found in them at any time over this period, and laboratory examinations confirmed the absence of this pest. The plants were of excellent vigour throughout, and they cropped well—in marked contrast to the untreated control plants in adjacent rows which came from an area where there was little eelworm. A small number of the treated plants were found to be infested with *Aphelenchoides* spp., and the control of these eelworms was therefore not quite complete.

Following this first trial, numerous other trials of the treatment for 7 minutes at 115° F. were carried out on stocks infested with the Stem eelworm in the Cheddar, Axbridge and Winscombe districts of Somerset. The varieties included Axbridge Early, Royal Axbridge, Bradley Cross, Huxley, Auchincruive Climax, Cambridge 448 and Royal Sovereign ; the dates of treatment ranged from August 12 to October 20. In all cases there was a good control of the eelworm, and loss of plants or serious effects on growth were found only where the planting was done badly, in very dry soil, or without proper firming.

As a result of all the trials it is concluded that the drawbacks found in the past to the hot-water treatment of strawberry runners may be overcome by shortening the period of treatment from 20 minutes at 110° F. to 7 minutes at 115° F., and that when this is done an excellent control of Stem eelworm may be obtained without adverse effect on the subsequent plant growth. For complete control of *Aphelenchoides* spp., 10 minutes at 115° F. may be necessary.

I am indebted to Mr. J. F. Southey and to Mr. J. Mayor for much assistance in this work, and take this opportunity of expressing my thanks to all those growers who kindly provided plants and ground for the field trials.

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PRELIMINARY EXPERIMENTS WITH METALDEHYDE SUSPENSIONS AGAINST SLUGS

by B. D. MORETON

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An investigation into the value of metaldehyde in spray form against slugs under field conditions was begun in 1951, following a preliminary report of the finding by Cragg and Vincent (1952) that metaldehyde was more toxic to slugs as a contact than as a stomach poison, and that slugs could be immobilized by contact with a 0·02 per cent aqueous solution.

Four types of spray material were used : a 0·02 per cent aqueous solution made by shaking metaldehyde with water ; solutions of higher concentration prepared by dissolving metaldehyde in methyl alcohol and then mixing with water ; suspensions of commercial powdered metaldehyde, further ground and mixed with water containing 0·1 per cent of a spreader, which with occasional agitation gave a suspension sufficiently stable for spraying ; and a suspension of a 25 per cent wettable metaldehyde powder. All the preparations were applied as fine sprays at the rate of 94 ml. per square yard, equivalent to 100 gallons per acre.

Three sets of field plots were arranged to test the effects of various concentrations of metaldehyde. A row of plots was marked out in a patch of grass and clover, each plot measuring 2 yards by 2 yards and being separated from its neighbour by a yard-wide untreated strip. The treatments shown in Table 1, replicated four times in randomized blocks, were applied on September 14. The weather remained cool and damp, and counts of slugs found on the surface were made the following afternoon. The majority were *Agriolimax reticulatus* Mull. with smaller numbers of *Arion hortensis* Fer.

TABLE 1

NUMBER OF SLUGS FOUND ON SURFACE ONE DAY AFTER TREATMENT ON CLOVER LEY, 1951

Metaldehyde Applied	Slugs Found in Three 1 sq. ft. Samples in each Block				Mean Number of Slugs per sq. ft.
	I	II	III	IV	
0·02 per cent aqueous solution ..	4	4	4	2	1·2
0·17 per cent in aqueous suspension	13	9	23	9	4·5
0·5 per cent in aqueous suspension	18	15	19	16	5·7
0·5 per cent in alcoholic suspension	24	8	16	28	6·3
Control (mean of ten 1 sq. ft. samples in untreated inter-plot strips)					0·6

The number of slugs found after treatment with the 0·02 per cent aqueous solution were significantly less than after the other three treatments, between which there was no significant difference. It was assumed that the slugs had been affected since they had not retired to shelter, but owing to the difficulty of confining them to sprayed surfaces, and at the same time giving them protection against birds but not the weather, no attempt was made to discover their ultimate fate.

The first results were confirmed in a further experiment, this time on bare soil, with plots measuring 2 yards by 1 yard and treatments replicated four

(57620) A**2

times. The " catch " three days after spraying was some ten times greater with the 0·5 per cent suspensions than with the 0·02 per cent solution. The addition of 0·5 per cent sodium pectate to the spray fluid resulted in a slightly, but not significantly, higher number of slugs affected. No data on persistence were obtained as there were few slugs in the succeeding three days.

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Control (untreated)	1	2	2	4	9

The number of slugs found after treatment with the 0·5 and 0·25 per cent suspensions were significantly greater than those with the 0·1 per cent, which was not significantly different from the controls. The number of lettuce plants destroyed was small, and though greater in the controls and with the 0·1 per cent treatment, the differences were not significant.

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With metaldehyde costing 16s. to 18s. per lb., the spraying method may prove uneconomic for such crops as cereals; in any case, in weather conditions when action against slugs is often demanded, the land is unsuitable for the passage of spraying machinery. The method is, however, promising for seed-beds and more valuable crops such as lettuce, strawberry and flowers under cloches and Dutch lights.

Field experiments are complicated by the dual action of metaldehyde: on the one hand, death caused directly by poisoning where concentrations are high enough; and, on the other, temporary immobilization by lower concentrations causing a break in feeding and exposing the slugs to the lethal effects of drying weather conditions. The ultimate effects of this temporary result are difficult to record, so that parallel observations on plant damage are useful. These effects will vary with weather conditions which will also influence the rate of evaporation, and thus the persistence of the metaldehyde.

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THE SHASTA DAISY MIDGE AND OTHER INSECTS IN FLOWERS OF *CHRYSANTHEMUM* SPECIES

by H. F. BARNES

Department of Entomology, Rothamsted Experimental Station, Harpenden, Herts

The recent occurrence of the gall midge, *Contarinia chrysanthemi* Kieffer, as a pest of Shasta Daisy in the Newcastle-on-Tyne area (Cohen, *Plant Pathology*, 1952, **1**, 51–3), made it desirable to know more about its life history and bionomics, its distribution on wild Ox-eye Daisy, and the possibility of its spreading to other cultivated chrysanthemums.

Although no systematic survey for the presence of this midge on the Shasta Daisy (*Chrysanthemum maximum* Raymond) was made during 1952, the larvae were detected in a sample of flowers from a garden at Humberstone, near Grimsby. They were also found on a plant from Woodthorne, Staffs, and in the flowers of the varieties Etoile d'Anvers and Esther Read in the National Botanic Garden, Dublin.

Some preliminary host-plant range tests using midges reared from the variety Esther Read were made in 1952. Females were observed to oviposit in the flower buds of the following *Chrysanthemum* species: *C. carinatum* (annual chrysanthemum), *C. cinerariaefolium* Vis. ("insecticide" pyrethrum), *C. frutescens* Thunb. (Marguerite or Paris Daisy), *C. leucanthemum* L. (wild Ox-eye Daisy) and also on Sweetheart chrysanthemum.

Emergence of the midge was obtained during late November and early December by keeping larvae from Esther Read in soil under room temperature in the laboratory from the beginning of August. Larvae kept in the unheated insectary started emerging on May 19, 1952, and continued until June 30. During May, 17 males and 72 females emerged, and in June 59 males and 130 females; the sex ratio thus being 27 : 73, a not unusual figure for *Contarinia* species of gall midge. There is a second, if only partial, emergence later in the year. Midges emerged between July 29 and September 8 from a fresh supply of larvae on Esther Read received from Newcastle on June 25, 1952. Midges also emerged between July 29 and August 28 from wild Ox-eye Daisy flowers received on June 6 from Anglesey. The possibility of such midges ovipositing on chrysanthemums in bud at this season of the year must not be overlooked.

GALL MIDGES FOUND IN WILD OX-EYE DAISY, 1952

Larvae of *Contarinia chrysanthemi* were found in 33 of 101 samples of wild Ox-eye Daisy flowers received in 1952. Their presence on this plant was established in Bedfordshire, the Isle of Ely, Berkshire, Buckinghamshire, Hampshire, Kent, Oxfordshire, Cornwall, Devon, Gloucestershire, Wiltshire, Anglesey, Caernarvonshire and Cardiganshire, and also in County Meath and County Wexford in Ireland.

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One or more species of the genus *Clinodiplosis*, whose larvae are usually inquilines, were reared from material from Huntingdonshire, the Isle of Ely, Berkshire, Oxfordshire, Devon, Wiltshire, Anglesey, Cardiganshire, Monmouth, Jersey and County Wexford. Larvae were present in samples from Bedfordshire, Hertfordshire, Buckinghamshire and Kent. *Lestodiplosis* species, whose larvae are predaceous, were reared from material collected in Yorkshire, Staffordshire, Derbyshire, Bedfordshire, Hertfordshire, Huntingdonshire, the Isle of Ely, Berkshire, Buckinghamshire, Hampshire, Kent, Oxfordshire, Cornwall, Devon, Gloucestershire, Worcestershire, Anglesey, Caernarvonshire, Cardiganshire, Glamorgan, Jersey and Aberdeenshire. Larvae were found in a sample from Shropshire. A species of the gall midge *Phaenobremia* was reared from samples received from Bedfordshire, Hertfordshire and Berkshire. The occurrence of this species may be considered to be accidental as its larvae feed on aphids.

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STEM EELWORM IN LUCERNE

by E. B. BROWN

National Agricultural Advisory Service, Cambridge

Damage to lucerne by Stem eelworm, *Ditylenchus dipsaci* (Kühn) Filipjev, is well known overseas, notably in the U.S.A., South Africa, New South Wales and on the continent of Europe. The evidence suggests that this is a specialized race attacking lucerne only, but López-Cristóbal (*Helminth. Abstr.*, 1946, 15, No. 236, 85) has reported that in the Argentine it may attack both alfalfa (lucerne) and oats.

The first report of the eelworm on lucerne in this country was in March 1948, when specimens of infested plants were received from Dunmow, Essex. Since then, four other cases have been investigated in the eastern counties, namely, at Little Wakering, Essex, in March 1949, at Ringstead, Norfolk, in May 1951, at Brockford, East Suffolk, in December 1951, and at Thriplow, Cambs., in April 1952. There have also been reports of damage to a crop in Kent in July 1948, and of extensive attacks on several fields in the Lindsey division of Lincolnshire in April 1949.

The symptoms are very similar to those of Stem eelworm on broad red clover. In the field small circular patches of stunted plants appear, the patches rapidly increasing in size as the attack progresses. Individual plants are dwarfed and exhibit swollen petioles which do not grow away. In clover the swollen petioles often form a rosette on the ground, whereas in lucerne there appears to be an increase in the number of shoots, which in a severely-damaged plant may form a mat close to the ground. Infested seedlings show characteristic swelling at the bases of the petioles (Plate I).

In attempts to obtain evidence on the possible source of the infestations in the eastern counties, it was found that the seed at Little Wakering, Brockford and Thriplow was wholly, or in part, of Hungarian origin—the source of the seed sown in the other two fields could not be traced. It was doubtful whether lucerne had been grown previously on any of the five fields ; certainly, it had not been grown on any of them for many years. As regards cropping with other legumes which might conceivably have acted as sources of infestation, at Dunmow there had been clover in 1933, vetches in 1935 and trefoil and wild white clover with ryegrass in 1939 ; the lucerne was sown in 1944. At Little Wakering, apart from clover in 1923 and 1933, there had been no legumes between 1920 and 1948, when the lucerne was sown. At Ringstead, where the lucerne was sown in May 1950, the only recent legumes were clover in 1938 and 1944. At Brockford, where the lucerne was sown in 1950, and extensive eelworm patches were present in December, 1951, there had been no recent legumes except clover in 1945. At Thriplow, self-sown plants of broad red clover, wild white clover and sainfoin were present among the eelworm patches, but they were all perfectly healthy.

If, as it appears, the pest is a specialized race of the Stem eelworm, affecting only lucerne, it seems certain that all the infestations originated with the seed.

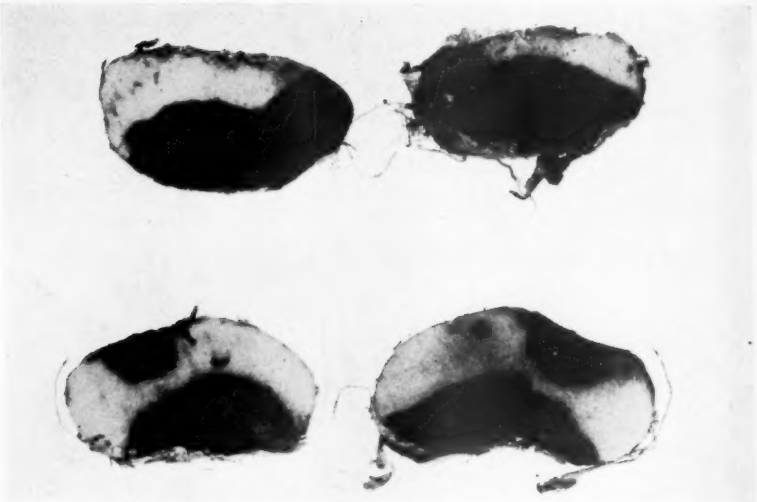
Thanks are due to Mr W. E. Dant for the photographs.

Above : A lucerne plant severely infested with Stem eelworm, showing swollen petioles and stunted growth, with healthy growth (*left*) for comparison. *Inset :* An infested seedling showing characteristic swelling.

PLATE I

(57620)

CORM ROT OF GLOXINIA (see p. 71)



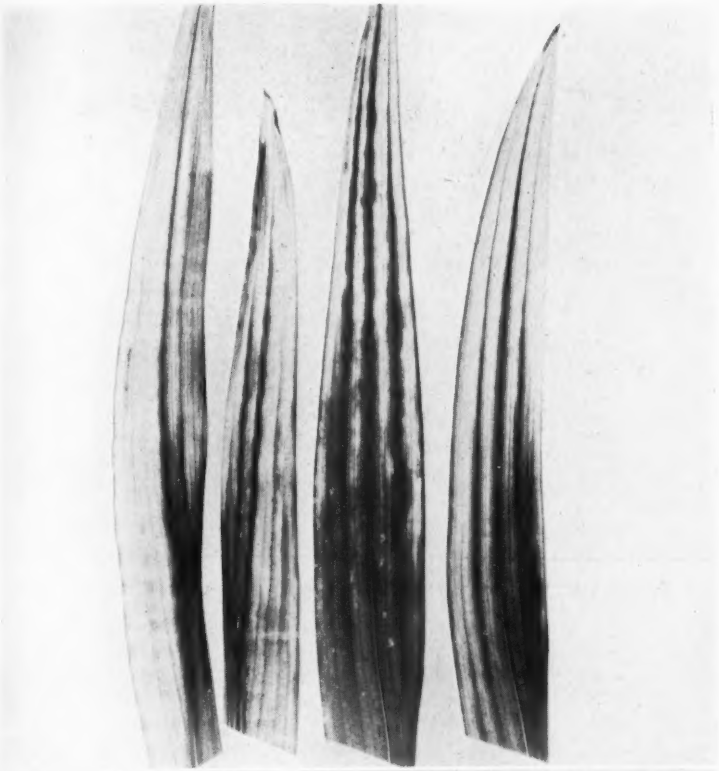
1. Sections of corms showing rotted and discoloured areas.

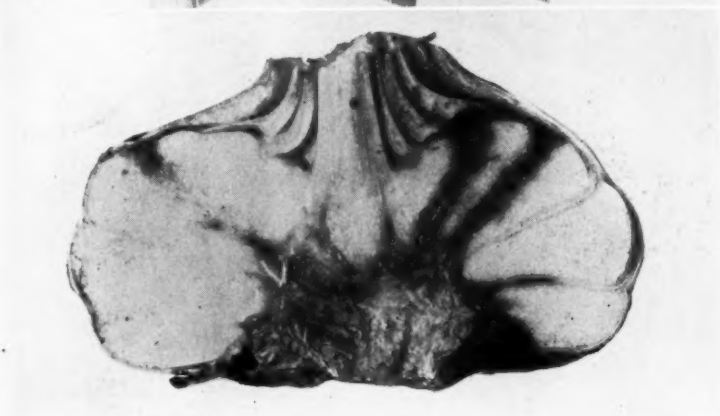
CENTROSPORA ACERINA ON CELERY (see p. 72)

2. Root rot in naturally infected plant.

3. Lesions on petioles fourteen days after inoculation.

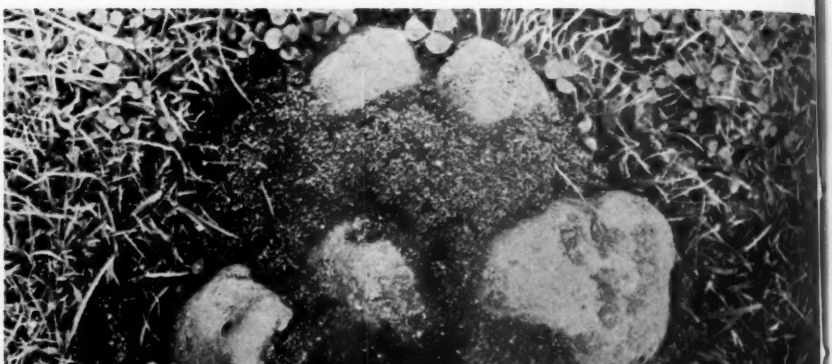
PLATE II





1. *Above :* Leaf tips of yellowed plants showing initial interveinal striping. Photographed by transmitted light.
2. *Below :* Section of corm showing diseased core and infected vascular strands leading to axillary buds.

PLATE III



1. Group of potato tubers, with cover removed, one day after being placed on an ant hill on a lawn, showing soil thrown up by the ants, *Lasius flavus* F.

2. Tubers showing ant damage. *Left, above :* One day after placing on ant hill ; *below :* After three days. *Right :* After seven days.

PLATE IV

INJURY TO POTATO TUBERS BY ANTS

by W. A. R. DILLON WESTON

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In August 1951, a sample of Catriona potatoes showing a peculiar form of pitting on the surface of the tubers at lifting, was sent to us by Mr. A. E. Cox of the National Agricultural Advisory Service, Essex, who had received them from a gardener growing the potatoes for exhibition. The pitting of the tuber surface was similar to that described and figured by Moore (1948), who noted that there had been a number of records of its occurrence on potatoes at lifting in various parts of the country during the years 1942–46, but that its cause was unknown. When a specimen of the potatoes from Essex was sent to the Plant Pathology Laboratory for examination, Dr. Joan Moore confirmed that the pitting was symptomatically the same as in the previous cases, and mentioned, *inter alia*, that there were some grounds for a belief that it might be caused by ants. S. G. Jary *in litt.* had expressed this opinion concerning a case at Wye, Kent, in 1943, but there was no corroborative evidence. When the possibility of ant damage was suggested to the grower, he thought it quite a likely explanation, as there were several nests of ants in the near vicinity of the potatoes and he had noted the ants at digging time.

While these specimens were under discussion I noticed some ants, subsequently identified as the common Mound ant (*Lasius flavus* F.), on a lawn at my home, and I took the opportunity of using them for an experiment. For this purpose healthy potato tubers, variety Majestic, were placed on each of three small ant hills, and transparent plastic cages were placed over them. Similar cages were placed over potatoes on ground where there was no visible trace of ants, but nothing of note happened to these tubers, and they showed no sign of injury during the course of the experiment. This was not so, however, with the tubers placed in the immediate vicinity of the ants, for when they were examined a day later, they were partially covered with soil (Plate IV, 1), and when some of them were lifted from the ant hill, depressed circular areas were observed on the surface that had been in contact with the soil (Plate IV, 2). The margins of the affected areas were well defined and the surface of the concave pits was smooth.

The symptoms were more acute the longer the tubers were left undisturbed. After three days the pits had increased in diameter, and in some of them the central part of the skin had broken away. After seven days many of the pits had coalesced to give large irregular depressions, still with well-defined margins, but with extensive breaking of the skin, due, apparently, to collapse of the underlying cells (Plate IV, 2).

While these experiments were in progress, Mr. E. C. Large told me that in March 1951 he had produced pitting very similar to that shown in Plate IV, 2 (top, left), at the site both of small pricks and of lenticels on the surface of healthy young tubers, by swabbing them or exposing them to the vapour of several chemicals, notably freshly-distilled formic acid, but also formaldehyde and ammonia. Subsequently, I injected some tubers with formaldehyde and obtained similar results.

The damage suffered by the potatoes in the ant hills closely resembled that in Pit Rot, first described by Pethybridge (1919) and so named by him because he had never seen it, even in an incipient stage, except in tubers on their removal from the " pit " or clamp. Subsequent experience seems to indicate that potato

tubers both in the ground and in store may suffer similar injury as a result of penetration of deleterious vapours or liquids. The present experiments show that ants must be included among the possible causal agents of such damage, but further observations are required to determine exactly how the injury is caused.

Thanks are due to Mr H. W. Wheeler for the photographs.

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POTATO SPRAYING TRIALS IN YORKSHIRE, 1947-51

by A. BEAUMONT, J. H. BANT AND I. F. STOREY

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While it has long been known that spraying with Bordeaux mixture is the best method of controlling Potato Blight (*Phytophthora infestans* (Mont.) de Bary), very little work has been done on its economic basis, that is to say, on ascertaining whether the cost of routine spraying, carried out over a number of years, is recovered by increases in crop yield resulting from the spraying. The aim of the present trials was to investigate this matter for the important potato-producing area round Goole, near the Humber, on the southern border of Yorkshire. In this area, blight appears as early as anywhere in Yorkshire, and the epidemic is generally more severe than in other parts of the county. If it is established that protective spraying is not economically justified in this area this may also be taken as true for the whole of Yorkshire.

EXPERIMENTAL METHODS

The variety used each year was Majestic, and the plants were generally at about A Certificate level as regards freedom from virus diseases. The individual plots were one-hundredth of an acre in area and they were arranged in two 3 × 3 Latin squares, the treatments being: (*a*) unsprayed control; (*b*) one spraying; and (*c*) two sprayings. Bordeaux mixture, 4 : 5 : 40, was applied at the rate of two gallons per plot with a knapsack sprayer. The first application was given at the end of July, and the second (for *c*) three weeks later. The actual dates of the applications in each year are given in Table 2.

The plots were examined at weekly intervals for blight and the observations were recorded according to the scale adopted by the British Mycological Society (1947). From these records, curves were plotted for the progress of blight in the sprayed and unsprayed plots in each year, and from these curves the " half decay " or " 50 per cent " dates were obtained (Fig. 1 and Table 2).

The plots were lifted at the end of September or beginning of October (Table 1). The middle five rows of each plot were dug and weighed on the same day, after sorting into ware, seed and chats by means of 2-inch and 1¼-inch riddles. The weight lifted in each plot ranged from 90 to 160 lb.

BLIGHT OBSERVATIONS ON THE HAULM

Table 1 shows the date of outbreak of blight (0·1 per cent by key) and the dates by which 50 per cent and 75 per cent of the foliage had been destroyed by blight, in the unsprayed plots, in each year of the trials. Previous observations in Yorkshire are in line with those in this table in indicating a rather late appearance of blight. In the period 1923–46, blight appeared in July in only seven seasons (1926, 1927, 1931, 1933, 1936, 1944 and 1945) and then mainly on early varieties. In three seasons the outbreak was delayed until September, and the remaining fourteen outbreaks occurred in August. At Goole in 1945, although the early outbreak was on July 11, the 50 per cent stage was not reached until August 30. In 1946 the date of outbreak was August 12, and the 50 per cent stage was reached on September 2.

TABLE 1

CHARACTERISTICS OF THE BLIGHT EPIDEMICS AT GOOLE, 1947–51.

OBSERVATIONS ON UNSPRAYED PLOTS

Year	Date of Outbreak	Blight at 50 per cent	Blight at 75 per cent	Date of Lifting
1947 ..	Aug. 5	Sept. 22	Sept. 28	Oct. 13
1948 ..	Aug. 13	Sept. 3	Sept. 6	Sept. 27
1949 ..	Sept. 1	Sept. 28	Oct. 1	Oct. 5
1950 ..	Aug. 4	Aug. 30	Sept. 2	Oct. 6
1951 ..	Aug. 28	Sept. 21	Sept. 28	Oct. 5

Not only is the Potato Blight epidemic normally late in beginning in Yorkshire, but also in many seasons the spread is slow in the first few weeks. For example, in 1947 blight appeared by August 5 but did not reach 5 per cent until September 9, and as noted above there was very slow progress in 1945. At the later stages of the epidemic the position is reversed and the progress after the 50 per cent stage is normally very rapid. In each of the years 1947–51 there was only a few days between the 50 and 75 per cent stages.

The full blight progress curves for both the unsprayed and the sprayed plots are shown in Fig. 1. The first spray application in each season was well timed in relation to the outbreak of blight, and so probably had about its maximum beneficial effect, as shown by Large (1945). Except in 1950 the amount of blight throughout the season was only slightly more on the once-sprayed plots than on the twice-sprayed, and only at the end of the season was the blight increase more rapid. The 1950 epidemic stood out as much more severe than the rest, and in this year the once-sprayed haulm was destroyed by blight significantly earlier than the twice-sprayed.

The protective effect of spraying on the haulm may be measured by the time interval in days between the 50 per cent points on the curves for the sprayed and unsprayed plots (Large, 1945). This " mean prolongation of growth due to spraying " in each of the years of the present trials is clearly shown in Fig. 1 and Table 2.

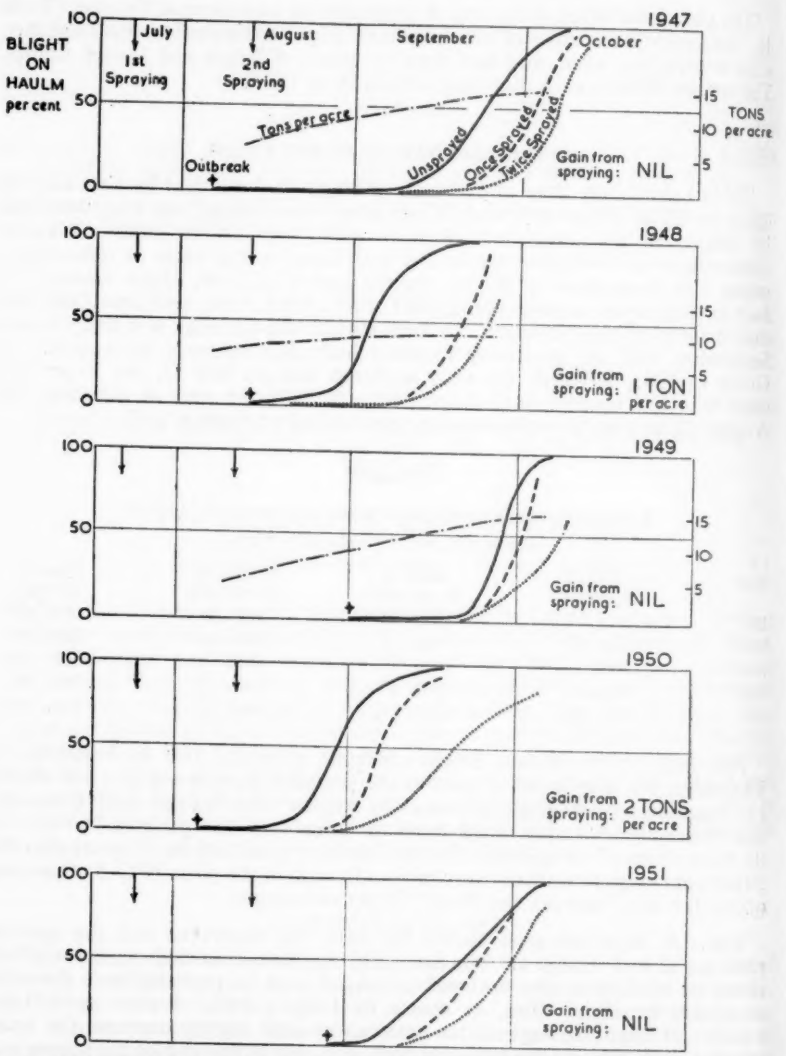

Fig. 1. Progress of blight on haulm of sprayed and unsprayed Majestic at Goole, Yorkshire, 1947–51. Growth curves for the tubers in the unsprayed plots are shown for the years 1947–49, and the approximate gain from two sprayings (tons per acre total crop) is given for each year.

TABLE 2

PROTECTIVE EFFECT OF SPRAYING ON THE HAULM

Year	Spraying Dates		Half Decay Dates (blight 50 per cent)			Prolongation of Growth by Spraying (days)	
			Unsprayed	*Once Sprayed*	*Twice Sprayed*	*Once Sprayed*	*Twice Sprayed*
1947 ..	July 22	Aug. 12	Sept. 22	Oct. 2	Oct. 6	10	14
1948 ..	July 23	Aug. 13	Sept. 3	Sept. 20	Sept. 24	17	21
1949 ..	July 21	Aug. 10	Sept. 28	Oct. 1	Oct. 8	3	10
1950 ..	July 24	Aug. 11	Aug. 30	Sept. 6	Sept. 19	7	20
1951 ..	July 24	Aug. 14	Sept. 21	Sept. 23	Sept. 28	2	7

CROP YIELDS IN RELATION TO BLIGHT ON HAULM

Murphy (1939) showed that tuber production ceased abruptly when the total weight of the haulm was reduced to about 53 per cent of its maximum. From his description of the state of the haulm this represents between 75 and 95 per cent blight on the B.M.S. scale. Large (1952) has provisionally taken 75 per cent as the point at which tuber production stops. It is unlikely that there is more than a very small amount of tuber production after that stage and this is offset by some reduction in the rate of tuber production between the 50 and 75 per cent stages.

Although the protective effect of spraying on the haulm is measured by the prolongation of its growth, the value of this prolongation, as regards increase of crop, depends essentially upon the time in the growing period at which it occurs. In the course of the present trials, sample crop liftings were made on several dates in 1947, 1948 and 1949, and the cropping curves, plotted in Fig. 1, serve to illustrate the fact, already well-established by Murphy (1939) and Doncaster and Gregory (1948), that the bulk of the crop increase takes place in August and becomes comparatively small in September, ceasing at the middle or end of September according to season. Hence, from consideration of the blight progress curves alone, it might be expected that there would be no gain from spraying in the years 1947, 1949 and 1951, but some gain in 1948 and 1950. It is of interest to note that from the average curve for Majestic already given in this JOURNAL by Large (1952), the probable gain in 1948 (75 per cent date September 6) would be estimated at about 9 per cent of the total potential crop, and in 1950 (75 per cent date September 2) at about 12 per cent. The actual yields obtained by lifting and weighing the crop are given in Table 3.

TABLE 3

EFFECT OF SPRAYING ON YIELD OF CROP

Year	Total Crop (tons per acre)				Ware (tons per acre)			
	Unsprayed	*Once Sprayed*	*Twice Sprayed*	S.E.	*Unsprayed*	*Once Sprayed*	*Twice Sprayed*	S.E.
1947	14·9	15·1	15·0	±0·25	11·8	11·8	11·5	±0·25
1948	10·3	11·6	11·3	±0·18	5·3	6·7	5·9	±0·17
1949	15·5	15·0	14·6	±0·57	12·7	12·2	11·2	±0·50
1950	13·6	14·6	15·7	±0·23	10·1	11·5	12·4	±0·27
1951	14·4	14·0	13·7	±0·20	12·8	12·3	12·1	±0·18

The weight of blighted tubers was negligible in all five years; only an occasional blighted tuber was found here and there in the sprayed and unsprayed plots, amounting to less than 0·1 per cent of the crop. The rows were always well banked and the tops were dead before lifting, except in 1948 and 1949 when the twice-sprayed plots were still green. This is a disadvantage of spraying as it keeps the foliage alive too near the date when lifting must be done.

It is well known that in some seasons, particularly in dry weather when there may be more than the normal amount of insect damage, copper fungicides can cause a certain amount of damage to the foliage, and it may be sufficiently severe to affect the crop. Some fairly severe yellowing and scorching due to this cause was observed in the two years 1949 and 1951, particularly in the twice-sprayed plots. This may be reflected in the lower yields from these plots, although the differences were not large enough to be significant.

As anticipated from the consideration of the curves for blight on the haulm, the increase in total crop due to two sprayings was nil in the years 1947, 1949 and 1951, about 1 ton in 1948 (just under 9 per cent), and about 2 tons in 1950 (just under 13 per cent). In 1948 the gain from one spraying did not differ significantly from that from two sprayings; and in 1950, the gain from one spraying was about half that for two sprayings—again in close conformity with expectations from the data for progress of blight on the haulm.

The economic indication from the trials over the five years is that even if commercial spraying were as well timed and as effective as the careful spraying done on the trial plots, and there were no loss due to damage by the sprayer wheels, or by copper injury to foliage after insect attack in dry seasons, the total gain from increase of crop by spraying over the five years 1947 to 1951 in the Goole area would have been only just sufficient to cover its cost. Protective spraying against Potato Blight is therefore scarcely justified as a routine measure on Yorkshire farms.

We wish to pay a special tribute to Mr J. Martinson, on whose farm the work was carried out, for his generosity in giving us full facilities, and for his great interest in the progress of the trials.

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THE FUSARIUM YELLOWS DISEASE OF GLADIOLUS

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Two major types of disease of gladiolus caused by Fusaria have been recorded in the literature. Massey (1926) described a Fusarium rot of stored corms which he attributed to *Fusarium oxysporum* Schlecht. var. *gladioli* Massey. In this disease, according to Massey's description, small reddish-brown, water-soaked spots may develop, chiefly on the sides and lower halves of the corm, by lifting time. During the storage period these spots increase in size and become more or less circular, rather sunken, ridged lesions, up to an inch in diameter. When slightly infected corms are planted, they give rise to weak plants which may die before flowering.

McCulloch (1944) described a " Fusarium Yellows " disease which she attributed to *Fusarium orthoceras* Woll. var. *gladioli* McCull. This disease is characterized by the yellowing of the foliage and collapse of the flower spike during the growing season. The fungus is readily isolated from the vascular core of the corm and also from lesions on the surface of the roots. In storage, internal rotting of the corm may develop. In Britain the only reference to similar diseases is that by Moore (1939) who mentioned the frequent occurrence in south-west England about 25 years ago of a disease of early-flowering gladiolus associated with a species of *Fusarium* not unlike *F. orthoceras* var. *gladioli*. Recent observations by Robertson (1951, 1952) have shown, however, that a disease of gladiolus plants in the field, very similar to Fusarium Yellows, does exist in Britain, especially in the Grandiflorus varieties, and it is responsible for some of the premature yellowing which troubles British growers. It can usually be distinguished without difficulty from yellowing resulting from attack by *Sclerotinia gladioli* Drayton and *Botrytis gladiolorum* Timmerm., as well as from the non-parasitic browning condition described by Moore (1939). The present article is intended to give a preliminary account of the status of Fusarium Yellows in Britain.

LEAF AND ROOT SYMPTOMS

In leaves of plants suffering from Fusarium Yellows the first signs of yellowing appear simultaneously in the terminal 3–6 inches of the outer leaves, where an interveinal pale green mottling changes rapidly to a bright yellow striping (Plate III, 1). This striped appearance progresses towards the leaf bases whilst the tips gradually become a uniform yellowish-brown ; meanwhile, the veins at the tip turn brown. In severe cases, the whole of the parts of the plant above the ground quickly die back. The degree of yellowing and the speed of die-back vary when different isolates of the *Fusarium* are used as inoculum. Slight differences in leaf symptoms have been observed on the different gladiolus varieties.

The gladiolus plant produces two kinds of roots during the growing season—fibrous roots and " droppers ". The former constitute the first signs of growth and are produced from the perimeter of the basal plate of the corm. Many side roots are produced from the main branches of this system, and the prolific growth which occurs provides for the plant until the young corm begins to form at the base of the flowering stem. At this stage, some sixty-five days after planting, the thick " dropper " roots grow out transversely from the new corm

and, following the contour of the old corm, grow rapidly downwards—their main function being that of re-adjusting the level of the young corm in the soil (Hartsema, 1937).

The symptoms of Fusarium attack are almost identical on the two types of roots, although on the " droppers " the lesions are rather larger, less frequent and mostly confined to the underside. Small light-brown lesions occur at all points along the root, most frequently at the tip and, later in the season, at the crown. Lateral growth of the fungus along the length of the root causes brown necrotic stripes usually half an inch in length but sometimes extending up to two inches. Girdling of the root occurs in the later stages, especially at the crown, and the cortex not infrequently sloughs off. The lesions become progressively darker in colour and are often quite black. A cortical rot extends inwards and is delimited by a dark brown zone of cells with thick dark walls, often filled with a brown resinous substance. Penetration of the thick-walled endodermis and, ultimately, invasion of the root vessels, is frequent but not general. Sections of infected root tissue show the presence of hyphae in the cortical and stelar cells ; fragments of surface-sterilized roots bearing the brown lesions invariably yield the *Fusarium*.

CORM SYMPTOMS

Both types of root are in direct vascular connection with the corm vascular tissues, and infection results from the fungus growing through the cortex and stele of roots into the corm base. Infected corms show, in the early stages, a blackened ring of root bases, when sectioned just above the basal plate. There is a marked discoloration of the vascular core (Plate III, 2), and the fungus is present both in the discoloured tissue and in the clean tissue in advance of it. The radiating vascular strands of the corm core may become dark brown and give rise to surface lesions. Young corms may become infected directly from the old corm through the vascular tissue or, probably less frequently, from diseased " dropper " roots.

We have been able to distinguish three major corm symptoms : internal browning with no external symptoms at first, but with ridged surface rot occuring at the nodes after storage ; ridged basal corm rot not affecting the vascular core (Massey's disease) ; and dry core rot of corms in storage, with no surface lesions except a dry hardness of the basal plate. *Fusarium* isolates from corms exhibiting each of these symptoms have caused yellowing.

INOCULATION EXPERIMENTS

Artificial inoculations have been made with various isolates of *Fusarium* from the corm core of yellowed plants taken from the field. The isolates were grown on 3 per cent cornmeal-sand, and, diluted with five times the volume of clean sand, added to pots containing unsterilized John Innes compost (Robertson, 1952). In 1950, symptoms similar to those already recorded for Fusarium Yellows were obtained in experimental work in which mixed isolates of *Fusarium* from corm lesions were used (Robertson, 1951). Later, in 1951 (Robertson, 1952), isolates from G. Hommerson plants showing no Fusarium Yellows symptoms in the field produced the disease under experimental conditions. In 1952, three isolates were used, two from the core of the young corm of yellowed plants and one from a ridged surface rot (Massey's disease) of a stored corm. The results, recorded on June 28, 1952, are given in the table on p. 63.

INCIDENCE OF FUSARIUM YELLOWS FOLLOWING INOCULATION OF CORMS, 1952

Source of Inoculum	Number of Plants Yellowed			
	Paul Rubens	*George Hommerson*	*Lavender Dream*	*Picardy*
Corm core of yellowed Wedgwood ..	2	13*	15	2
Corm core of yellowed Polynese ..	10	15	3	1
Ridged surface rot of George Hommerson corm	8*	14*	15	1
Controls, not inoculated (15 of each) ..	1	1	2	0

* Out of 14 plants inoculated ; all other figures are for 15 plants inoculated.

It is evident from this table that a fungus which is capable of causing a ridged surface rot is also capable of causing symptoms similar to those of Fusarium Yellows when infection takes place through the soil. The variety Paul Rubens showed resistance to the *Fusarium* isolated from a yellowed plant of the variety Wedgwood ; Lavender Dream showed resistance to an isolate from the yellowed Polynese variety ; Picardy was resistant to all three isolates.

IDENTITY OF THE FUSARIUM

Examination of selected isolates from the collection of 150 taken from corms and roots of yellowed plants has suggested that there may be a range of *Fusaria* within the morphological boundaries of *Fusarium orthoceras* var. *gladioli* (McCulloch, 1944) and *Fusarium oxysporum* var. *gladioli* (Massey, 1926) which can cause yellowing. This hypothesis is based on the observations of such characters as macroconidial measurements, ability to produce sporodochia, pionnotes and sclerotia, colour variations on different media, etc. *Fusaria* isolated from corms of yellowed plants grown at Cambridge were stated by Dr. W. L. Gordon, of the University of Manitoba (*in litt.*), to be morphologically indistinguishable from his types of *Fusarium oxysporum*. Furthermore, Snyder and Hansen (1940) have already suggested that *Fusarium oxysporum* can embrace many biological forms previously given taxonomic status by Wollenweber. Dr. Gordon (*in litt.*) has suggested that *Fusarium orthoceras* var. *gladioli* may probably be regarded as a special form of *Fusarium oxysporum*.

It is suggested, therefore, that the organism causing the disease known as Fusarium Yellows of Gladiolus be incorporated in *Fusarium oxysporum* Schlecht f. *gladioli* Snyd. & Hans., as described by Snyder and Hansen (1940), from the point of view of a classification based on morphological features.

We do not wish, at this stage of the investigation, to draw conclusions regarding the relationships of Massey's *Fusarium oxysporum* and McCulloch's *Fusarium orthoceras* from the point of view of pathogenicity. It is clear, however, from our investigations, that isolates from Massey's type of corm rotting can cause symptoms of McCulloch's type of yellowing. Nelson (1948) has already stated that there is a difficulty in distinguishing between isolates from the two types of disease, and that he uses the reaction of the variety Picardy in differentiating between the two diseases, for this variety, which is resistant to McCulloch's type of Fusarium Yellows, is very susceptible in storage to the ridged surface rotting.

DISTRIBUTION

The disease has been found in the field in Holland by one of us (E.W.B.), and it has also been found in corms exported from Holland to Britain. Since so many of the corms grown in Britain are of Dutch origin, the disease might be expected to be widespread here. In fact, we have found the disease among all the large stocks of gladiolus that we have examined in this country, including a number in Ayrshire, Cambridgeshire, Cornwall, Midlothian and Surrey.

In the course of the present investigation we have found no fewer than 33 varieties to be frequently affected by Fusarium Yellows in Britain. Others, such as Picardy and Bit of Heaven, have shown resistance.

CONTROL

As the fungus can be carried over in the vascular core of the corm, treatments with surface disinfectants are ineffective. There is good evidence that much of the spread of the disease is by means of corms already infected internally. It has been shown (McCulloch, 1944 ; Robertson, 1952) that warm conditions favour symptom expression. In America the fungus has been shown to persist in the soil for long periods, but it is not clear how important infection from the soil is in Britain. In our experience, stocks of gladiolus varieties vary in the amount of disease they show, in a way which suggests that the origin of the stock is of great importance. It seems that, in the absence of expensive soil treatments, corms grown for sale can be kept relatively free of the disease, if healthy stocks are obtained in the first place, by careful roguing and adequate rotation. Many of the newer varieties appear to be susceptible, but sufficient resistance is present in existing stocks of gladiolus to enable resistant varieties to be produced if required.

We are indebted to Dr. W. L. Gordon for generous help with the taxonomy ; and to Mr. F. T. N. Elborn for the photographs.

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TRIAL OF AMMONIACAL GAS LIQUOR AGAINST POTATO ROOT EELWORM

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In May 1951 it was suggested that this department might test the action of ammoniacal gas liquor on Potato Root eelworm, *Heterodera rostochiensis* Woll. In view of the shortage of sulphur, sulphuric acid and ammonium sulphate, the direct use of ammoniacal liquor as a nitrogenous fertilizer had been envisaged, and it was desirable to forecast the effects of such treatment on Potato Root eelworm. The substance had been tested before in field plots, notably by Holton (1938) and Miles, Henderson and Miles (1943). Holton's results were evaluated by yield of potatoes only, and were thought satisfactory because on the untreated plot the plants made only slight growth and finally died, while on the plot treated with liquor at the rate of 9,680 gallons per acre there was a good yield of large potatoes free from taint. Miles, Henderson and Miles used 4 × 4 Graeco-Latin square designs at four centres, using four potato varieties with untreated controls and three chemical treatments (one of them gas liquor at the former high rate) at each centre in 1938. In 1939 the same four treatments were repeated using one potato variety throughout, and in 1940 the four varieties were tested with no chemical treatment. Here the criteria were both yield of tubers and numbers of viable eelworm cysts per 10 c.c. of soil; some significant yield increases were obtained, but the differences in eelworm densities were neither significant nor consistent from year to year.

CRITICAL POT TESTS

A technique for the testing of nematicides under the controlled conditions of a pot test had been developed at Rothamsted (Peters, 1952), and it was decided to apply this method for the testing of the gas liquor. A feature of the technique is the simultaneous testing of several materials using one batch of mixed infested soil in cylindrical glazed pots each holding 20 lb. But since the 1951 tests were already in progress, it was necessary to run the gas liquor test separately and in clay pots of comparable volume. It is interesting that such highly significant results nevertheless came from so small an experiment. The statistical methods employed for evaluation of the eelworm criteria have already been fully described (Peters, 1952), and they will not be repeated here.

The pots, each containing 20 lb. of well-mixed soil moderately and uniformly infested with eelworm cysts, were treated in the following ways :

A. With 500 ml. of water only (control).

B. With 100 ml. of gas liquor and 400 ml. of water (equivalent to 1,800 gallons of gas liquor per acre).

C. With 500 ml. of gas liquor (equivalent to 9,000 gallons of gas liquor per acre).

The gas liquor, kindly supplied by the Luton Gasworks, contained about 0·9 per cent free ammonia by weight and 1·4 per cent total ammonia. The specific gravity of the liquor was 2° Twadell. In point of total application of ammonia, irrespective of its form, the 1,800 gallons of gas liquor would be equivalent to about 8 cwt. sulphate of ammonia per acre, and the 9,000 gallons to 40 cwt. There were three replications of each treatment. One month later,

on June 28, 1951, the soil in each pot was thoroughly mixed and sampled for eelworms ; one healthy seed tuber of the variety Arran Banner was then planted in each pot. The height of the plants after six weeks was measured for evidence of phytotoxicity or plant stimulation.

During the following winter the crop of tubers in each pot was harvested and weighed and the soil was again sampled. In each of the soil samples, at planting and at lifting, the mean number of eelworm cysts per 200 grammes of air-dry soil was estimated ; the mean number of larvae hatching per cyst in potato root diffusate was also estimated using two batches of 100 cysts each, following the methods described by Peters and Fenwick (1949). The number of cysts per gramme of soil, multiplied by the number of larvae hatching per cyst (i.e., the number of hatched larvae per gramme of soil) was taken as a measure of the soil infestivity. The results are given in Table 1.

TABLE 1

RESULTS OF POT TRIALS OF AMMONIACAL GAS LIQUOR FOR THE CONTROL OF POTATO ROOT EELWORM, 1951

	Treatment per 20 lb. of Soil		
	A. 500 *ml.* water only	B. 100 *ml. gas liquor* and 400 *ml. water*	C. 500 *ml. gas liquor only*
Height of plants at 43 days (*cm.*)	27·3	28·3	35·0
Weight of tubers produced (*grm.*)	42·3	84·3	347·3
Mean weight per tuber (*grm.*)	9·6	17·6	24·7
Yield increase, percentage of control ..	—	99	821
Eelworm cysts per gramme of soil :			
At planting	0·79	0·98	0·78
At lifting	4·90	4·70	9·80
Number of larvae hatched per cyst :			
At planting	88	87	57
At lifting	162	200	283
Infestivity of soil :			
At planting	69	85	45
At lifting	797	948	2,776
Percentage kill	—	1·4	34·8

The results showed no evidence of any phytotoxic effect of the gas liquor, even at the higher dosage, the height of the plants in the treated soil being greater (but not significantly greater) than in the untreated. There was a large and highly significant increase both in size and total yield of tubers due to the treatments.

The figures for cysts per gramme of soil at planting indicate a moderately heavy infestation such as might frequently be met with in the field, and there was no significant variation in the density of cysts among the pots. There was a significant reduction at planting in the number of larvae hatching per cyst from the high-dosage pots, indicating a significant kill of 35 per cent with gas liquor at the rate of 9,000 gallons per acre. But in practice such a kill would be too low to be of much value because the survivors multiply on the succeeding crop at a higher rate on treated than on control plots, partly owing to reduced competition.

The figures for eelworm infestation at lifting are of importance. The cyst density showed a sixfold increase in the controls, which is not unusually high. The cyst density with gas liquor at the rate of 1,800 gallons per acre was not

significantly different from that in the control pots; but at the rate of 9,000 gallons per acre there was a twelvefold increase, which was significantly higher. With this treatment the number of larvae hatching from the cysts was so much higher than in the controls that the cysts must have been larger or better filled with eggs. The overall effect of the increases in number of cysts and larvae hatching per cyst was to increase the soil infestivity figure from 797 in the controls to 2,776 with the higher dosage of gas liquor. The soil infestivity at the end of the experiment with treatment at the higher dosage was forty times greater than that of the untreated soil at the start.

CONCLUSIONS

The conclusions are that ammoniacal gas liquor used at the rate of 1,800 gallons per acre in pot tests had negligible effect on both the potatoes and the eelworms. At the rate of 9,000 gallons per acre it led to a greatly increased yield of both tubers and eelworms.

It is dangerous to forecast from pot tests what the performance of nematicides may be in field trials, for conditions in the field are always more variable and the materials are nearly always less efficient. But in the present trials the pots were maintained out of doors, plunged to the rims in gravel, so that conditions were not too artificial. So far as it goes, this experiment suggests that it would be unwise to use ammoniacal liquor as a source of nitrogen for potatoes on eelworm-infested land, as the increased yield (whigh might well result) would be purchased at the cost of greatly increasing the eelworm population. If some other crop took the fertilizer effect it is probable that the eelworm population would remain rather lower than in the controls; on the other hand, the potato crop is usually the most valuable one in a rotation and farmers would naturally wish it to receive the fertilizer benefit.

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DELAYED SPORULATION OF *PHYTOPHTHORA INFESTANS* ON INFECTED POTATO SHOOTS

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Various workers have found *Phytophthora infestans* (Mont.) de Bary developing on sprouts from blighted tubers. Early work was reviewed by Salmon and Ware (1926) and observations since then have been summarized by Peterson (1947). There is little information on the history of the fungus between the time of emergence of the diseased sprouts and the development of the disease on the adult plant. Because of this, the view has sometimes been held that sprout infection is insufficient to account for the outbreaks later in the season. The experiments recorded here show that the fungus can grow for months, without sporulating, within the developing tissues of the host, and that it will not spore until the environmental conditions become suitable.

EXPERIMENTS WITH KING EDWARD TUBERS, 1949–50

Fifteen King Edward tubers were inoculated with the field strain of *P. infestans* in mid-December 1949, and planted in pots of sterilized soil in a glasshouse in mid-March. Only seven tubers sprouted. On one plant a necrosis of the stem at soil level was noted after a month, and though this necrosis advanced up the stem and the lower leaves withered, the plant continued to grow, and on May 24, sixty-nine days after planting, it was 4 feet high. The plant was then placed in a humid atmosphere and five days later the stem and leaves were covered with the sporangiophores of *P. infestans*. On another plant a basal rot appeared in June. This plant also continued to grow, but the lesion dried up, and when the plant was put in a humid atmosphere at the end of August no *P. infestans* developed. The remaining five plants showed no symptom of disease.

During the same season twelve King Edward plants were raised in pots and, when 7 inches high, were inoculated by inserting pieces of tuber or leaf tissue, parasitized by mycelium of *P. infestans*, into cuts in the stems, the wounds being bound round with adhesive tape. Necrosis rapidly appeared near the point of inoculation on all plants, and four months later, at the end of August, the stems were either dead or severely discoloured, with many dying leaves. There had been no sign of fructifications during the four-month period, but when the infected tissues were put in a damp atmosphere the fungus developed on portions of them. This experiment, and that described above, indicated that the fungus could develop in the growing tissues of the host for very considerable periods without fructifying.

EXPERIMENTS WITH CANSO TUBERS, 1951–52

Further experiments were made in 1951–52 using the Canadian variety Canso. In the late summer of 1951 Canso became blighted in the field at Rothamsted, and when the fungus was isolated it was found to be a B strain. Samples of apparently healthy and obviously blighted tubers were received from Rothamsted and twelve of the latter were potted in November 1951, the pots being kept in a glasshouse. On one of the plants a basal necrosis was observed in January, thirty-nine days after planting, and the leaves were becoming yellow ; four days later the necrosis had extended 4 cm. up the stem from soil level. When

the stem was put in a damp atmosphere, abundant *P. infestans* developed upon it. In March, four other plants developed a basal necrosis which gradually extended upwards. On three of these, when they were put in a damp atmosphere, an abundant growth of *Botrytis cinerea* developed. *P. infestans* was, however, recovered from the fourth plant. The basal necrosis on this plant was first observed on March 8 ; by March 26 the necrosis extended upwards for 22·5 cm., and a second shoot showed basal necrosis and wilting. When the wilted shoot was removed and put in a humid atmosphere, a rich growth of sporangiophores developed upon it. Transference of sporangia to a panel of differential hosts identified the fungus as a B strain. On the stem, which was still growing, the necrosis extended steadily upwards ; a leaf wilted, was incubated and *P. infestans* spored on the petiole. By April 10 the necrosis had reached a height of 31 cm. ; another leaf wilted and, when exposed to a high humidity, the fungus fructified on the petiole. On April 14, four-and-a-half months after planting, the necrosis extended to 36 cm. from soil level and the whole plant was put in a humid atmosphere. Five days later, the necrosis had reached a height of 39 cm. and *P. infestans* was sporing at this level.

These observations corroborated those made upon the inoculated King Edward tubers. As the plants were under continual observation and as in the whole experience of our glasshouses no plant has become spontaneously blighted in the early months of the year, it seems certain that the *P. infestans* which eventually spored on tissues well above soil level developed from the mycelium originally present in the tubers.

In the middle of April a further ten of the naturally infected Canso tubers from Rothamsted were planted in large pots of sterilized soil, as were three apparently uninfected tubers. The pots were put in an outside open frame and later moved into the open. The pots were not in contact with the ground and were isolated between two glasshouses so that they were not immediately adjacent to any potato plant. All three of the supposedly blight-free tubers sprouted, but only five of the blighted ones. All eight plants were sturdy and appeared completely healthy at each weekly inspection until July 23, when they had begun to show signs of maturity. Between this date and mid-August necrosis appeared on the stems of all five plants grown from blighted tubers, and *P. infestans* was recovered from the tissues of all when they were incubated in a damp atmosphere. During this period there was no sign of any blight in a neighbouring field of potatoes. On some of the stems the necrosis was basal ; on others it was at some distance up the stem, but when such stems were split open they were found to be discoloured internally to the base. On two stems the necrosis was faint, yet, when incubated, *P. infestans* developed along the whole length of one and in patches along the other. On two other stems the fungus developed upon still unfolded leaves at the growing point, and on one plant it spored on a stolon. On one plant there were four stems showing necrosis to various levels, and *P. infestans* spored upon all. Sporangia from one stem were identified as B^2 strain.

The pots were brought into the laboratory in mid-August and the tubers harvested at the end of the month. In one pot, five out of eleven tubers had a blight-like rot. These five tubers were sliced and incubated ; *P. infestans* developed and was identified as a B^2 strain.

When the fungus was found to spore on the stems of plants grown from blighted Canso tubers, the plants grown from apparently blight-free tubers were examined. Only two of these were still available and they were maturing.

All the stems were split open and incubated. On one plant, one stem had basal necrosis and another internal necrosis, and upon these the fungus spored. As this plant had been brought into the glasshouse in early June, where it is extremely unlikely that it would have picked up aerial infection and where it would certainly have been noted had it done so, it must be presumed that the original tuber was infected, though this infection was not apparent when the tubers were inspected at Rothamsted and by me on their receipt and at the time of planting. The behaviour of the fungus in this plant is circumstantial evidence that the infection in the plants outside originated from the tubers and not from air-borne inoculum. It would also seem unlikely that infection arose from the soil, all of which was steam sterilized, or from the water supply, as no blighted potato plant or tuber has been found in the glasshouses during the 1952 season.

DISCUSSION WITH REFERENCE TO PREVIOUS WORK

An experiment similar to those here described was carried out many years ago by Massee (1906). He grew plants from blighted tubers : three in a warm, dull, humid glasshouse, and three in a cool, well-lighted, dry house. The former plants " showed the first indication of *Phytophthora* when the shoots were six weeks' old, and a fortnight later the three plants were blackened and destroyed by the fungus ". The latter " showed no trace of disease at the end of two months, when one of the plants was removed to the warm house and placed under a bell jar. Within nine days this plant was blackened and killed by the fungus ". Two weeks later a second plant behaved similarly. McAlpine (1911) also held the view that the mycelium grew up into " the stalks and foliage, where the spores are produced ".

Massee's experiment and his conclusions were severely criticized by Pethybridge (1911), who repeated the experiment with proper controls and found no evidence in support of Massee's theory that blight epidemics were caused by dormant mycelium. Pethybridge summed up his findings thus : " There is no evidence at present existing to show that the attack of the potato crop as a whole with blight occurs otherwise than by aerially borne " spores," if we except the few diseased plants from diseased tubers which do undoubtedly occasionally occur, but which can scarcely be regarded as being part of the general crop ". In the present experiments there is, of course, no proof that the fungus would have spored on the infected plants grown outside had there been " blight weather." It thus remains unproven that such plants can act as foci of infection in the field, though there seems every likelihood that they would do so. What has been proved is that, under certain conditions, *P. infestans* can remain alive for long periods within the growing tissues of the plant without sporulating.

I thank Miss B. S. Rogers for technical assistance.

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CORM ROT OF GLOXINIA

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During the past twenty-five years complaints have occasionally been received about an unexplained rot of Gloxinia corms in storage. In the early months of 1951 such rotting was unusually prevalent and occurred in many stocks imported from Belgium, though it was not always apparent on arrival in this country. In one instance a high proportion of 7,000 corms, which left Belgium on January 10, was found to be decayed on reaching Manchester, nine days later : in another the corms appeared healthy when they arrived in December 1950 and the rot did not develop until after storage for several weeks.

Samples from different batches showed the same symptoms. The affected corms, though soft, could not readily be distinguished externally from the healthy ones. When cut, two kinds of symptoms were observed. In some corms the internal tissues were soft, wet, and pink, yellow or chocolate in colour, or they developed such coloration within a few minutes of cutting. In others there were one or more distinct dark brown lesions extending inwards from the surface, and sometimes both symptoms were present in the same corm (Plate II, 1). Isolations from the discoloured tissues gave negative results, but those from the dark brown lesions invariably yielded *Cylindrocarpon radicicola* Wollenw., and on occasion also *Pythium de Baryanum* Hesse. Inoculations into healthy corms with these two fungi were made at the Plant Pathology Laboratory, but the results were negative except once, when an extensive dark brown lesion was induced in one of six corms inoculated with the *Pythium*.

In most years little is heard of the trouble. It caused some concern, however, in 1936, when most of the specimens received showed a dry, corky, basal rot, as though the affected tissues were drying up. Indeed, in some instances the diseased portions could readily be removed. No parasitic organism was found in the tissues and many corms started to grow, and produced normal plants when laid out in a moist, warm greenhouse on coconut fibre.

The cause of Corm Rot is still uncertain. Gloxinia corms are known to be very sensitive to changes of temperature, and it is generally believed that they react unfavourably to chilling. For instance, some growers are convinced that the basal rotting can be induced merely by placing the corms on a cold floor. The disease may, in fact, be primarily a non-parasitic trouble due solely to unfavourable conditions of storage. Or it may be caused by a fungus acting as a primary parasite, or as a secondary organism which cannot invade the corms unless some predisposing factors have been at work. The need for a careful study of the trouble is clearly indicated.

NEW OR UNCOMMON PLANT DISEASES AND PESTS IN ENGLAND AND WALES

***Centrospora acerina* on Celery.** In late November 1952 patches of slightly stunted yellowish plants were noticed in several crops of celery at Maulden in Bedfordshire. The soil is an organic sandy loam and in some parts of this area celery has been grown continuously for many years. Similar symptoms had been seen in previous years, the effects being most severe in those crops which had been left in the ground until early in the New Year. The specimens examined showed rotting of the roots spreading to the stem and base of the petioles (Plate II, 2). The dark green or black colour of the older lesions was very characteristic. When cut open the roots showed a brown discoloration of the vascular cylinder. Mycelium developed readily when pieces of rotten tissue were placed in water, and after some days it produced spores typical of *Centrospora*. *Pythium* sporangia were also found on many of the young roots. Cultures from the root tissue yielded reddish to black colonies with a characteristic toruloid mycelium, from which spores were obtained by placing pieces of agar from the edge of a colony in water. The fungus was identified as *Centrospora acerina* (Hartig) Newhall. The *Centrospora* was inoculated into apple fruits and produced a rot from which the fungus was re-isolated. Mature celery plants were also inoculated, and after ten days a rot developed (Plate II, 3); again the fungus was re-isolated ; conditions of high humidity were required for this rotting to take place. This is the first record of the *C. acerina* occurring in this country. It causes a serious storage rot of celery in the United States and Canada where Truscott (*Canad. J. Res.*, 1944, **22**, 290–304) regards the disease as affecting senescent plants only. It may well be that attack by *Pythium* or some other fungus rendered the crops at Maulden susceptible to attack by *Centrospora*.

I. F. STOREY AND H. J. WILCOX

Cereal Root Eelworm on Grasses in the West Midlands. In April 1950, at Broseley, Salop, cysts of a species of *Heterodera* were found on the roots of cocksfoot, *Dactylis glomerata* L., which had followed a crop of oats attacked by Cereal Root eelworm, *Heterodera major* (O. Schmidt). In 1951 plants with cysts on the roots were found in a four-year-old stand of seed cocksfoot at Middleton, near Ludlow, Salop, and on experimental plots in a field at High Hatton, Salop, where oats had been seriously affected by Cereal Root eelworm, cysts were found on perennial ryegrass (*Lolium perenne* L.), Italian ryegrass (*Lolium italicum* A. Br.), cocksfoot, meadow fescue (*Festuca elatior* L.) and timothy (*Phleum pratense* L.). Although no biological tests were made, the cysts were carefully examined and are believed to have been *Heterodera major*. Fewer cysts were found on timothy than on the other grasses. With the possible exception of cocksfoot, the grasses did not appear to show visible symptoms above ground. Cysts of *Heterodera* spp. do not appear to have been previously recorded on cocksfoot ; all the remaining grasses were recorded hosts of *H. schachtii* before the separation of *H. major* as a distinct species, and the species found on these grasses was in all probability *H. major*. Cultivated and wild grasses may help to maintain, or even to increase, soil populations of the eelworm in the absence of cereal crops. Following a partial failure of oats, a field in this province was put down to a ten-year ley. When it was ploughed and oats were taken again, the crop was visibly affected by Cereal Root eelworm.

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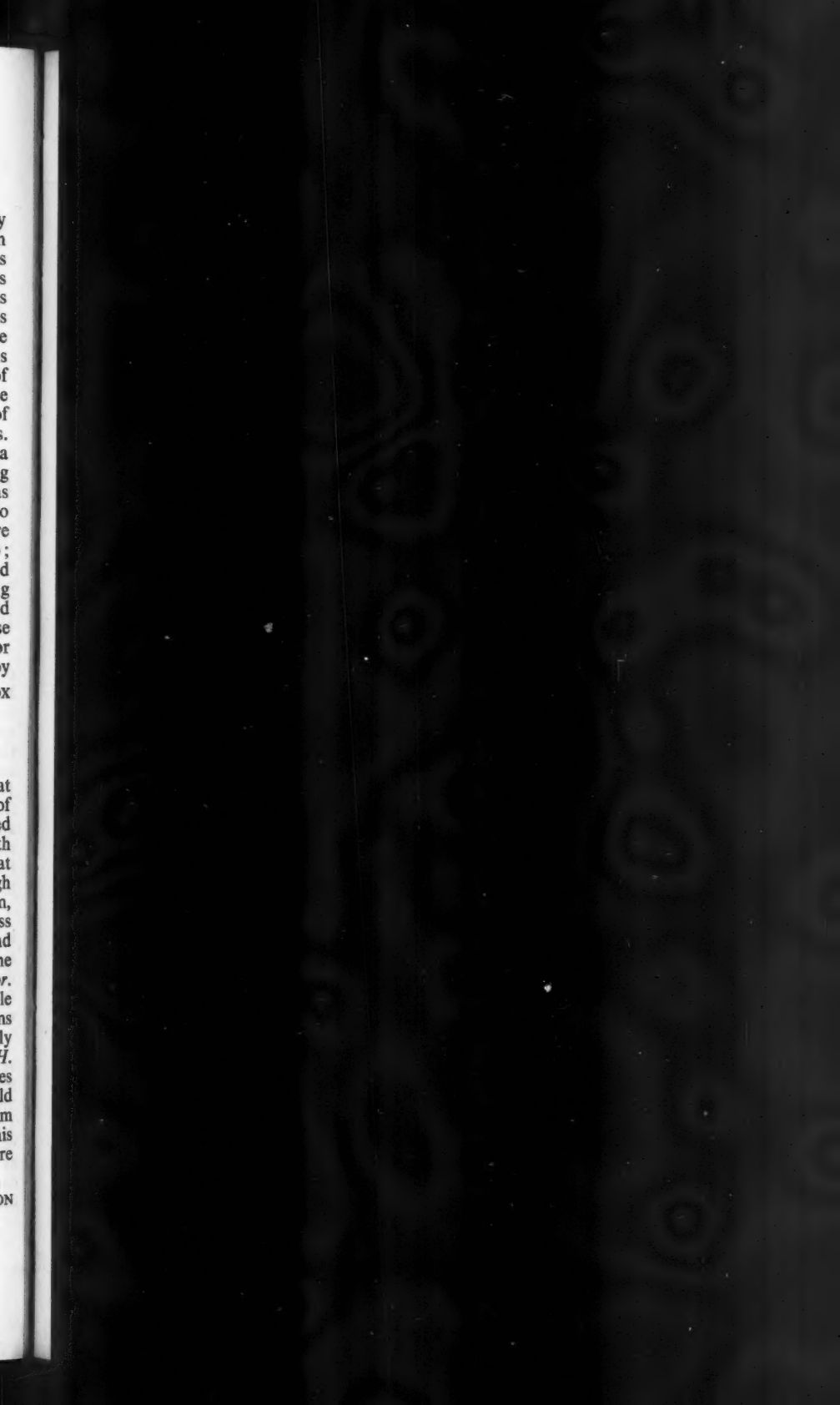
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